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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

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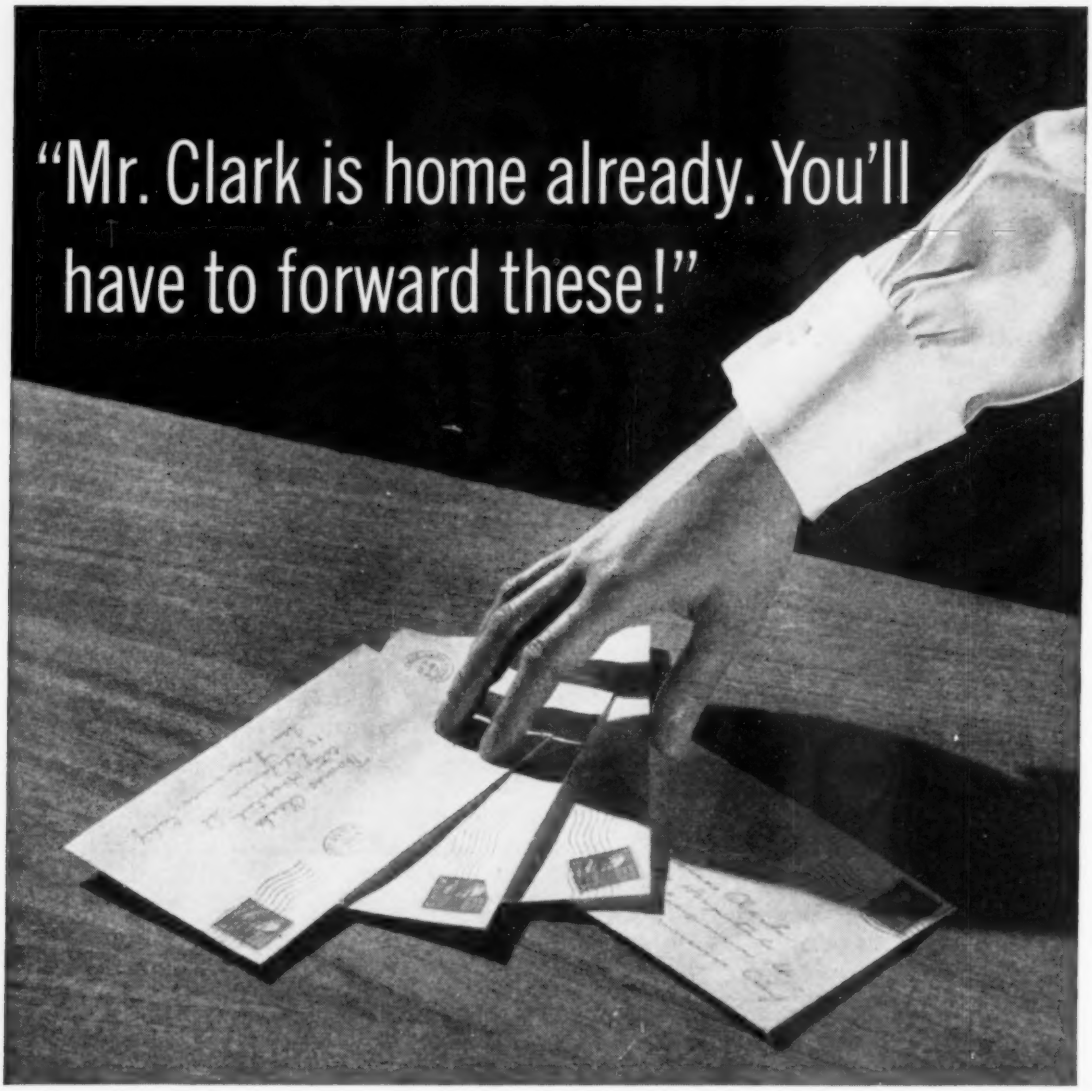

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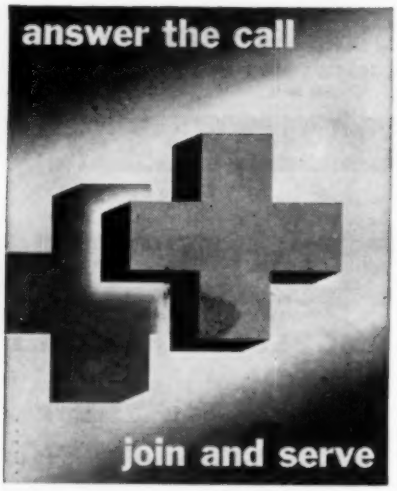

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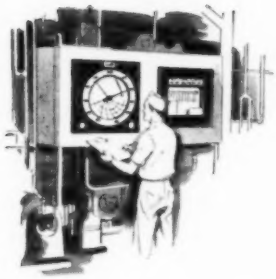

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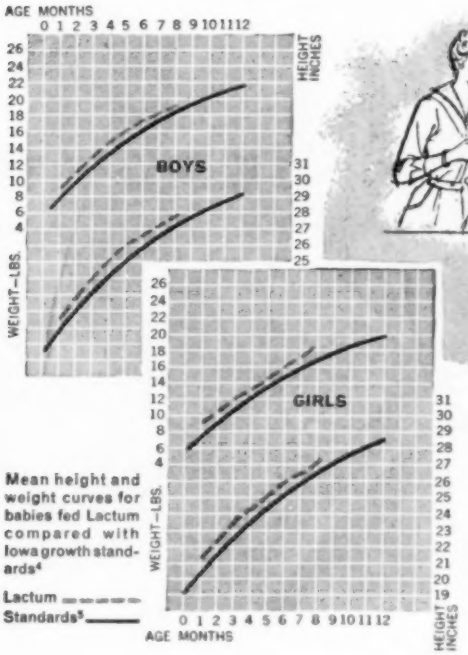

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VOL. 71 JUNE 1954 No. 6

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HEREDITY IN PROGERIA*

WITH FOLLOW-UP OF TWO AFFECTED SISTERS

ABDEL HAMID MOSTAFA, D.Ch., M.R.C.P.

AND

MAMDOUH GABR, M.B., D.Ch., M.D.

Cairo, Egypt.

Progeria, or Hutchinson-Gilford disease, is considered to be one of the rarest affections met with in childhood. Only 22 typical cases were described in the literature till the present time. Since a comprehensive review of the literature was recently reported by one of us (Gabr, 1954) a short account of the syndrome will be given.

The disease usually starts at the end of the first year of life by loss of hair and arrest of growth, both in height and in weight. Gradually the typical features of the disease set in with alopecia, loss of eyebrows and eyelashes, prominent eyes, small face, receding chin, overcrowded teeth and dwarfism. Due to loss of subcutaneous fat, the limbs look thin with relative enlargement of the joints together with limitation of their movement, hence the stooping attitude of the patient. The hands are specially affected, the nails atrophied and transverse, the skin thin and the fingers flexed, looking like the hands of an old person. The alopecia and the stooping attitude of the patient give the child the characteristic elderly appearance. As the child grows older, arteriosclerotic

*From the Pediatric Department, Kasr el-Aini Faculty of Medicine, Cairo, Egypt. This is a follow-up of the article, "PROGERIA," which was published in the February 1954 Archives of Pediatrics. The presented cases are the first familial ones of this syndrome to be reported.

changes set in and the patient usually dies of coronary thrombosis at the second decade of life. Coxa valga, small clavicles and thin bones are always evident on x-ray examination. Defective ossification of the skull is also commonly present. Other investigations are usually normal.

All the reported cases of the disease were sporadic. The following two patients are the first familial cases to be described.

CASE REPORTS

Family History. A complete history of the family was difficult to obtain, but Chart 1 represents available data.

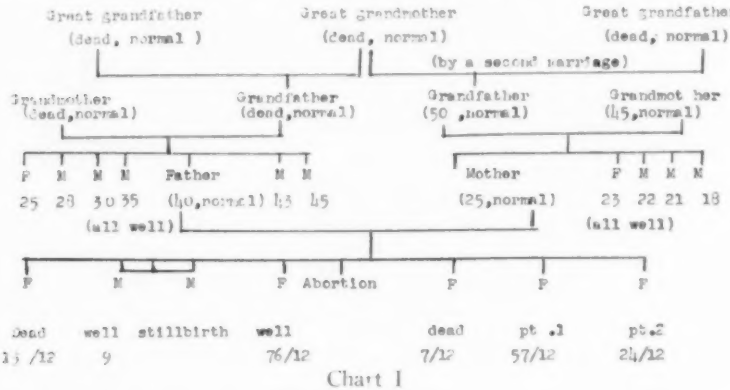

Chart 1

The parents of the present patients were first cousins. The mother gave birth to 6 children; 2 died from intercurrent infections at one year three months and seven months, respectively. She had one stillbirth (a twin) and one abortion. The two patients are the youngest members of the family. No history of any dwarfism, alopecia, absent clavicles or presenile changes was present in any member or relative of the family. Both parents and all children were examined by us and were found to be normal in appearance and measurements. None showed any sign related to the disease.

Case 1. A. M. was four years old when first seen on September 20, 1952. Her condition at that time was fully reported before by Dr. Gabr (1954).

A brief account of her condition at that time follows: The disease started with alopecia when she was seven months old.

Arrest of growth was noticed by the mother after the first year of life. When examined at that time the patient presented typical features of the disease. Her height was 76 cms. and weight 8.25 Kgms. Her skull circumference was 46.5 cms.; the fontanels and cranial sutures were open. Her scalp hair, eyelashes and eyebrows were almost completely lost. Her mandible was small and receding, and the lower teeth were overcrowded. Only the sternal ends of both clavicles could be felt. Her posture was bent, due to the flexed attitude of all her joints, which were enlarged. The subcutaneous fat was lost except over the pubic area and cheeks. The skin was thin, icthiotic, and a sclerodermic element was present, especially over the hands which looked elderly with the fingers flexed and nails atrophied and transverse. The apex was in the fourth space midclavicular line. Her blood pressure was 108/65. X-ray examination revealed: coxa valga, absent clavicles, thin skull bones with separation of sutures and a persistent anterior and posterior fontanels, E.S.R. was high, 60 mm. per hour.

The girl was readmitted to hospital because of whooping cough on April 20, 1954 when she was five years and eight months old. Her measurements were as follows: Height 84 cms.; weight 8.850 Kgms; skull circumference 47 cms. Her general appearance did not show marked change, except for an increase in her stooping attitude and more limitation of joint movements. The anterior fontanel was still open for one inch, the posterior fontanel was half an inch in width and the sutures were all felt separated. The few fine hairs over the scalp and at the site of the eyebrows were still present, but the eyelashes were completely lost. The lower mandible was receding, and the lower set of teeth were overcrowded. Her lower canines were absent. A fine brownish pigmentation was apparent over her lower abdomen and upper thighs, but the skin otherwise showed little change from the previous examination. It was thin, icthiotic, and sclerodactylic with atrophied transverse nails apparent in the fingers and toes. Her blood pressure was 110/60. The clavicles were rudimentary, only one centimeter could be palpated at the sternal end, the rest being replaced by a thin fibrous cord. Investigations did not reveal marked changes since the previous examination. There was a slight lymphocytosis as a result of the whooping cough. X-ray of the skull, pelvis, vertebral column were the same. An ossification

centre appeared in the carpus in addition to the three centres which were present in the previous examination. The Wassermann was positive once. The electrocardiogram did not show any abnormality.

Case 2. N. M., when first examined with her sister on September 20, 1952, presented few features suggestive of the disease and was briefly referred to with her sister's case report (Gabr, 1954). When re-examined by us on April 20, 1954, the full blown picture of the disease became apparent and a full description of her condition on both dates will be given.

Past History. She was a full term breast-fed infant, looking normal at birth. At the age of 7 months she could sit unsupported. Her first tooth erupted shortly afterwards. She could utter a few words at the age of one year and six months, and she seemed brilliant for her age but a little ill-tempered and irritable. The mother noticed loss of her hair ever since she was 5 months old. Arrest of growth was noticed by the mother when the child was one year old, growth in height and in weight almost ceasing since that time.

Examination on September 20, 1952 revealed a small infant weighing 5.5 Kgms; height 69 cms., skull circumference 39 cms. Apart from a partial alopecia, her head and face looked normal and proportionate with normal eyelashes, eyebrows, lower jaw and teeth. The anterior fontanel was felt open for one inch, but the cranial sutures could not be felt. The posterior fontanel was almost closed. Her clavicles were normal, measuring 5 cms. The skin was dry over the extensor surfaces of the upper limbs, but no icthiosis, pigmentation, loss of subcutaneous fat or wasting were present. Her fingers and toes were normal. The heart, lungs and liver were free.

X-ray examination of the skull showed a persistent posterior fontanel, but no separation of sutures (Fig. 1a). Radiological examination of the pelvis revealed slight coxa valga. The wrists showed three ossification centres. Her clavicles were well formed (Fig. 3a), and her spine was normal. Other investigations were within normal limits.

The patient was re-examined with her sister on April 20, 1954, when two years and four months old—she too suffered from whooping cough. The great similarity to her sister's condition was evident (Fig. 5) and marked progeric changes were noticable.

She could not stand unsupported. Her height was 72 cms; weight 6.800 Kgms., skull circumference 43.5 cms. The anterior fontanel was open for one inch; the coronal, sagittal and lamboid sutures were all felt to be separated for half a centimeter. The posterior fontanel was persistent, being one inch in breadth. She had lost most of her scalp hair, but a few fine hairs were still present on the vertex and temples. Shining dilated veins were present over the scalp. The eyebrows and eyelashes were very scanty. Her nose was peaked, her ears had small lobules. Her face looked small in relation to her head, with a markedly receding chin. The teeth of both jaws were overcrowded; the lower two canines were missing and the lower two medial incisors were overiding. The first and second milk molars were present. The mouth and the tongue were small, and the palate highly arched. The eyes were slightly protuberent, but otherwise normal. The skin over the

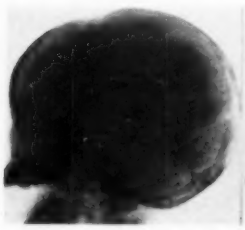

Fig. 1 a. Patient N. M. Skull (lateral) on Sept. 20, 1952.

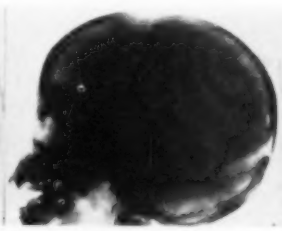

Fig. 1 b. Patient N. M. Skull (lateral) on April 20, 1954.

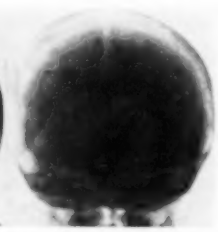

Fig. 2. Patient N. M. Skull (anteroposterior) on April 20, 1954.

body, especially over the abdomen and extensor surface of the upper and lower limbs, was very dry and sclerodermic in appearance. No pigmentation or icthiosis was present. There was a moderate loss of subcutaneous fat, but the buccal and pubic pads of fat were present. The skin changes were very evident on the hands, which looked very similar to her sister's hands, senile, and sclerodactylic, with flexion of fingers, present. The nails were all atrophied and more transverse than longitudinal (Fig. 6). The elbow joints looked enlarged, and extension of the elbows was limited, but movements of the lower limbs were free. The neck was short, the thyroid gland was normal. The clavicles were present, but small, measuring 5 cms. (normal for her age 7.5 cms.). The abdomen looked distended and the umbilicus was protuberent. An examination of her internal organs revealed no

abnormality. The chest was free. The apex was in the fourth space at the midclavicular line, her blood pressure was 95/60. The liver and spleen were not felt. The central nervous system was free, and the lymph glands, all over the body, were of normal size.

X-ray examination of her skull revealed persistence of the anterior and posterior fontanels and marked separation of the lamboid suture and to a less extent of the sagittal and coronal sutures. The pituitary fossa was normal. The maxilla and mandible were small with overcrowded teeth (Fig. 1b and 2).

The clavicles were characteristically small on x-ray examination, being visible only in their middle portions (Fig. 3b). The pelvis showed marked coxa valga (Fig. 4). The limbs showed generalized osteoporosis. There were three carpal ossification centres. The terminal phalanges were absent. The vertebral column was normal.

Investigations: Blood count revealed a marked lymphocytosis from the whooping cough: 76 per cent hemoglobin, R.B.C. 4,500,000, W.B.C. 27,000, lymph, 69, polys. 31. E.S.R. 38 cm. Blood calcium 13 mgm. %; blood phosphorus 5 mgm. %; blood urea 20 mgm. %; blood cholesterol 200 mgm. %; Wassermann reaction negative. Urine and stools were free. Electrocardiogram was normal.

Table 1. *Measurements of Both Patients*

Patient	Wt. Kgm.	Normal	Ht. cms.	Normal	Sk. cms.	Normal
Pt. 1. (Sept. 20, 1952).....	8.25	16.400	76	103	46.5	49.5
Pt. 1. (April 4, 1954).....	8.850	20.000	84	113	47	50.7
Increase	0.600	3.600	8	10	0.5	1.2
Pt. 2. (Sept. 20, 1952).....	5.500	7.250	69	67	39	43
Pt. 2. (April 4, 1954).....	6.800	13.400	72	91	43.5	48.5
Increase	1.300	6.150	3	24	4.5	5.5

COMMENT

The follow-up of the older patient (Pt. 1) did not reveal significant changes in her condition. It is apparent from Table 1, that her growth in height and weight were very slow in comparison to the average normal rate. Apart from the appearance of skin pigmentation over the abdomen and an increase of the sclerodermic change, she did not show appreciable difference in the 19 month's follow-up period. There was no clinical or electro-

cardiographic evidence of arteriosclerotic changes developing. The positive Wassermann, which was present, is apparently a coincidence and not related to the disease.

The development of the full-blown picture of the disease in the younger sister brings up the total number of reported typical cases to twenty-three. Due to the presence of the disease in the older sister, the minor changes in the younger patient, when nine months old, attracted our attention. This allowed us the unique opportunity of examining the younger patient at that early stage and following-up her condition until the full blown picture of the disease became established. It is apparent from Table 1 that the patient was of normal height at the age of nine months and did not show any appreciable growth in height since that time. Her weight increased by only 1.300 Kgms, during the same period in contrast to a normal increase of 6.150 Kgms, in healthy children. This delay of growth in weight and height after the first year of

Fig. 3 a. Patient N. M. Clavicles on Sept. 20, 1952.

Fig. 3 b. Patient N. M. Clavicles on April 20, 1954.

Fig. 4. Patient N. M. Pelvis (coxa valga) on April 20, 1954.

life is a feature of all reported cases of progeria. (Thomson and Forfar (1950), and Cooke (1953).) The skin changes were the earliest features to appear in this patient. The face, mandible and teeth were normal at the age of nine months. The prognathios gradually developed since that time, most probably due to arrest of growth of the mandible with resultant overcrowding of the teeth and typical facies.

The most striking feature in the follow-up period, however, was evident on x-ray examination. The only cranial abnormality that was present on the first examination was a persistence of the posterior fontanel (Fig. 1a); the sutures were normal. The second radiological examination (Fig. 1b), 19 months later, revealed wide separation of all cranial sutures indicating that this defect developed gradually as a result of arrested growth of the bones of

the vault in spite of the increase in the size of the brain (the skull circumference increased from 39 cms. to 43.5 cms.). The clavicles also showed characteristic changes. At nine months of age they were normal in size and shape (Fig. 3a). When the patient was re-examined, 19 months later, the clavicles were relatively small (measuring 5 cms., the average normal being 7.5 cms.) and in the x-ray they appeared slender, casting a shadow only in their middle third (Fig. 3b). These changes may be due to arrested growth of the clavicles alone or in association with some resorption of osseous tissue. The mild coxa valga, which was present on the first examination, became well developed on the second examination (Fig. 4).

Fig. 5. Patient A. M. and sister N. M.

Fig. 6. Hands of both patients.

The cranial defect and short clavicles present in both reported cases are features of cleidocranial dysostosis. This association of cleidocranial dysostosis and progeria is not rare and was present in three other reported cases. (Thiers and Nathan, 1933; Broc et al., 1935; Hughes, 1950). In fact prognathios, short clavicles and dwarfism, which are features of all the reported cases of progeria, are also features of cleidocranial dysostosis. It thus seems that a definite relation between the two diseases exists. The cranial bosses and scapular changes, commonly present in cleidocranial dysostosis, however, were not present in our reported cases. Furthermore, coxa valga was found to be more common in the described cases of cleidocranial dysostosis rather than coxa valga, which is present in all reported cases of progeria (Fitchet. 1929).

The above presented cases are the first typical cases of progeria with a familial incidence. It is apparent, from the family history,

that the disease was transmitted as a recessive characteristic. Since the patient's parents were paternal first cousins, it is apparent from Chart 1 that the pathological gene was originally transmitted from the great grandmother to the two grand parents and hence to both mother and father of the present patients. Progeria thus seems to be a recessively inherited disorder at least in the described family. The following facts are to be mentioned in this respect:—

1. Paterson, in 1922, described a progeric-like syndrome in two brothers with mental deficiency, the parents of whom were first cousins. The cases, however, were not typical cases of progeria. (Thomson and Forfar, 1950). Neil and Dingwall also described a syndrome resembling progeria in two mentally defective children (Neil and Dingwall, 1950).

2. Cleidocranial dysostosis, which is not rarely associated with progeria, is almost always familial in nature (Anspach and Heupel, 1939).

3. Werner's syndrome, so-called progeria of adults, a disease having several features in common with progeria of childhood, is a strongly familial disorder (Thannhauser, 1945).

4. A thorough review of the literature shows that the mother of the first case of progeria described in the literature (Hutchinson, 1886) had alopecia. Also the father of Thomson and Forfar's case was short and had a slightly receding chin.

The above evidence suggests that an hereditary element is to be considered in progeria. The sporadic nature of the disease in all the previously reported cases may well be due to the rarity of the syndrome and the absence of consanguinity in all reported cases (Thannhauser, 1945). It must be admitted, however, that until more familial cases are described, it is safer to state that progeria is sometimes transmitted as a recessive inherited characteristic.

The occurrence of heredity, the association with cleidocranial dysostosis, are in our opinion points against speculating an endocrine derangement to be the cause of the disease. Until more knowledge can be obtained it must be considered to be due to a congenital germ plasm defect which may be hereditary and which begins to manifest itself towards the end of the first year of life.

SUMMARY

Two sisters affected with progeria are reported. The develop-

ment of the various manifestations of the disease was closely followed-up in the younger sister.

The reported cases are the first typical familial cases of progeria described in the literature. It is concluded from the evidence given that progeria is sometimes transmitted as a recessive inherited characteristic.

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NEW FORM OF NIEMANN-PICK DISEASE. (Archivos del Hospital Universitario, Havana, 4: 1, Jan.-Feb. 1952). Osseous lipoidosis was found in two children, aged 4 and 5 respectively, with areas of osteolysis in the skull. Biopsy showed that foam cells were present in each case, and morphological and microchemical studies led to the conclusion that the material overloading the cytoplasm of the cells was a lipoid containing cholesterol. The chemical reactions resembled those described in Niemann-Pick disease (although the symptomatology was entirely different), and abnormalities were noted in the metabolism of vitamin A and the carotenoids. A single massive dose of vitamin A (2,400,000 international units in one case and 2,000,000 in the other) resulted in rapid improvement in the osteolytic areas; radiological evidence of cure was not obtained until later. One patient was also given vitamin A in doses of 180,000 units daily until a total of 12,200,000 units had been given. Both patients were cured. The therapeutic effect of vitamin A in these cases may be due to inhibition of the esterases that synthesize sphingomyelin or to an increase in the esterases that catabolize and oxidize the lipoids. The authors do not believe that the disease results from vitamin A deficiency, although such a deficiency or a change in the metabolism of the carotenoids may favor its development. They consider it to be a new form of osseous lipoidosis and have given it the name of osseous Niemann-Pick disease.—*Journal A.M.A.*

A NEW SPECIFIC TREATMENT FOR PERIANAL DERMATITIS

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Perianal dermatitis is due to fecal irritation and is characterized by an area of erythema, desquamation, superficial ulceration and exudation around the anus. The area involved may be several or more centimeters and will vary with the intensity and duration of the irritation. It is one of the most common eruptions encountered in newborn infants (sore bottom) and is frequent in infants and children. Until Pratt's pioneer description[1], perianal dermatitis was included with other rashes about the buttocks under the general term of diaper rash.

In the newborn it is apparently caused by transitional stools. Perianal inflammation is particularly annoying to the nursery personnel, since mothers invariably place the blame for the rash on poor nursing care. After the first weeks of the neonatal period, the dermatitis usually subsides spontaneously. In recent years, with the increased frequency of diarrhea and the use of oral antibiotics, and their attendant loose stools, perianal dermatitis has become more frequent in children of all ages and not uncommon in adults[2].

The purpose of this study is to ascertain the efficacy of an anti-enzymatic and antibacterial ointment containing a 1:1000 quaternary ammonium compound with a casein competitive substrate in a cod liver oil, water-repellent base (Diaparene® Peri-Anal).

PLAN OF STUDY AND RESULTS

A total of 250 newborn infants, under two weeks of age, was studied as follows:

1. *150 Cases with Prophylactic Care against Perianal Dermatitis:*
   50 cases with Diaparene Peri-Anal.
   50 cases with just soap and water.
   50 cases with vaseline base only.
2. *100 Cases with Perianal Dermatitis:*
   50 cases treated with Diaparene Peri-Anal.
   50 cases treated with soap and water.

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Table 1. *Treatment—100 Cases Alternated.*

	Diaparene Peri-Anal Treatment of Dermatitis	Soap & Water Treatment Peri-Anal Dermatitis
1. Area of Dermatitis	2 - 5 cm.	2 - 5 cm.
2. Duration of Treatment	2 - 3 days	4 - 6 days
3. Reaction to Treatment	None	Irritability -9 Pain & Crying -8
4. Recurrence	None	6
5. Stools	2 - 6 daily Meconium Soft-Yellow	2 - 6 daily Meconium Soft-Yellow
6. Comments	Ease of application	Saline Rinse used after soap and water

Table 2. *Prevention—150 Cases Alternated.*

	Diaparene Peri-Anal Prophylaxis	Soap & Water Prophylaxis	Vaseline Prophylaxis
1. Occurrence of Peri-Anal Dermatitis	1	8	6
2. Area of Dermatitis	2 cm.	2 - 4 cm.	2 - 4 cm.
3. Duration of Treatment	5 - 6 days	5 - 6 days	5 - 6 days
4. Reaction to Treatment	None.	Discomfort	None
5. Stools	2 - 6 daily Meconium Soft-Yellow	2 - 5 Meconium Soft-Yellow	2 - 5 Meconium Soft-Yellow
6. Comments	No Diaper Stain	Soap & Water followed by Saline Rinse After each BM.	Vaseline difficult to remove completely.

(All newborn infants received approximately the same diaper change care by the nursery personnel).

3. *Allergy:* Patch tests were made on 100 infants. They were left on for 72 hours, removed for one week and reapplied for 72 hours. There was no evidence of perianal sensitivity noted. In all the infants, where Diaparene Peri-Anal was used routinely, there was no evidence of hypersensitivity.

There were 100 cases alternately treated with Diaparene Peri-Anal® and soap and water (See Table 1). The extent of inflammation was from 2 to 5 cms., around the anus. All were moderately severe in degree, except for five cases which were very severe with beginning ulceration. None was given ultraviolet ray lamp exposure, or any other care, other than prompt diaper changes. Frequency of bowel movement was about the same in both groups, averaging from two to six daily.

All in both groups, responded to treatment eventually. Those treated with Diaparene Peri-Anal® required but half of the period necessary for cure with soap and water. Six of the controls had recurrence, despite prophylactic regimen of soap and water cleansing after each bowel movement.

The 150 prophylactic cases were alternated with: (1) Diaparene Peri-Anal®, (2) Soap and water and (3) Vaseline (See Table 2.). The prophylaxis in each case was continued throughout the newborn's hospital stay, namely, five to six days. Perianal dermatitis developed in 8 of the cases treated with soap and water and 6 of those on vaseline; one of the newborns treated prophylactically with Diaparene Peri-Anal® developed perianal dermatitis.

In the entire series of 100 infants (50 treatment; 50 prophylaxis), who received Diaparene Peri-Anal®, there was no systemic toxicity, local sensitivity or irritation.

Bacteriological tests with Diaparene Peri-Anal showed a 2 mm. zone of inhibition by agar cup plate method against micrococcus pyogenes var. aureus.

COMMENT

Although the association with diarrheal and frequent loose stools is clear, the etiology and pathogenesis of perianal dermatitis have never been satisfactorily elucidated. It has been suggested that perianal dermatitis may be due to increased alkalinity of the stools.

However, in a series of fifty consecutive cases of perianal dermatitis, both acid and alkaline stools were noted by us with approximately equal frequency.

The type of feeding does not seem to have any marked relationship to the dermatitis. Change in the feeding formula from evaporated milk to homogenized whole milk has been shown to be ineffective in preventing perianal dermatitis. Pratt reported only an 8 per cent drop in incidence by prescribing lactose in place of dextri-maltose® in a formula of cow's milk and water fed to 253 white infants. Hatfield, Simpson and Jackson[3], in a study of 180 healthy newborns fed various evaporated milk formulas, cow's milk formulas and human milk with water and dextri-maltose® added for equal caloric content observed; "It is our opinion that the low incidence of irritation of the buttocks noted in the out-patient group was due to the meticulous diaper care by the mothers."

A popular hypothesis for the cause of perianal dermatitis in children and adults, following oral antibiotics, is overgrowth of monilia organisms in the intestinal tract[2]. In their recent study of over 300 patients with perianal inflammations following oral antibiotic therapy, Manheim and Alexander[2] point out: "Inasmuch as exacerbations occur for such long periods of time, we must conclude that other factors are contributing to the prolongation of the condition. The most striking characteristics of this syndrome are its tendency to recur and its tenacity. . . . This has constituted a therapeutic problem of great magnitude."

We have suggested that in diarrhea and loose stools following oral antibiotics and transitional stools of the newborn, there is an altered enzymatic composition which produces an irritating effect on the sensitive skin around the anus. This then goes on to erythema, desquamation and ulceration. Secondary infection may further aggravate the rash.

Resistant cases of severe perianal irritation in infants and very small children have been reported as nonresponsive to the common local treatments. While some investigators report cortisone has been found to be of therapeutic value in topical treatment of "idiopathic" pruritus ani, it has not provided benefit in treatment of child and adult perianal inflammations following oral antibiotics[9].

Perianal dermatitis may be confused with other rashes in the diaper area:

1. *Ammonia Dermatitis* is characterized by an erythema in the diaper area, especially where contact with urine has been present for long periods. The rash may become papulo-vesicular and finally ulcerative. These lesions may become secondarily infected. In circumcized infants there is often an associated ulceration of the external urinary meatus with recurrent crusting and bleeding; in rare instances stricture may follow necessitating meatotomy. The pathogenesis of ammonia dermatitis is the production of ammonia in the voided urine by urea-splitting bacteria which are normal inhabitants of the intestine and which contaminate the skin about the buttocks and genitalia. The odor of ammonia in the wet diaper, especially in the morning, is pathognomonic. Ammonia dermatitis can be prevented by the use of a non-volatile bactericidal diaper rinse. Methylbenzethonium Chloride (Diaparene® Chloride) has been proven effective for this purpose[6] and has replaced the use of bichloride of mercury and boric acid which are potentially poisonous[7, 8]. This antiseptic in a water-miscible base (Diaparene® Chloride Ointment) may be used as an aid in the treatment of ammonia dermatitis[10, 11].

2. *Contact Dermatitis* (*Dermatitis Venenata*) Contact dermatitis is the term applied to rashes caused by contact of the skin with vegetable and mineral irritants. In the case of rashes about the diaper area, the dermatitis is caused by either soaps or lotions applied to the skin directly, or by chemicals used in washing, soaking or bleaching of the diapers. The newer detergents are said to be a frequent source of trouble.

Home automatic washers often fail in completely removing detergent residue from the laundered diapers. A diaper service which employs a "non-detergent" wash formula, preferably with an antiseptic rinse, such as diaparene chloride, may prevent this type of contact dermatitis.

The rash is at first maculopapular and limited to the diaper region, but then becomes exudative and spreads to the rest of the trunk. It is usually very itchy and frequently infected from persistent scratching. Ammonia dermatitis may be superimposed, aggravating the rash. The treatment is primarily to remove the irritating agent and the application of a mild water-miscible ointment.

3. *Intertrigo*—commonly referred to as chafing. This condition

is frequent in infants and children. It occurs where two moist skin surfaces are in apposition, such as the groin and inner thighs, buttocks, flexor surfaces of joints and neck. It is more commonly seen in obese children. Wet and fecal soiled diapers, which are allowed to remain on the infant for relatively long periods and lack of cleanliness, are contributory factors. If untreated, it may become infected. Occasionally, eczema may be superimposed on the erythema. There is local heat, marked erythema, exudation and soreness. The treatment consists in keeping the affected areas clean and dry. Frequent application of a mild antibacterial dusting powder and separation of the apposing surfaces with cotton rolls is often helpful.

4. *Atopic Dermatitis* is an allergic skin reaction due to the ingestion of certain foods. In the acute form, the lesions are characterized by erythema, pruritis and oozing. In the chronic form, the skin is dry and scaly and there is a tendency to crust formation. The rash usually involves the face and folds of the elbows and knees, but may occur on any part of the body. When it occurs in the diaper area, it may be confused with perianal dermatitis or any of the other dermatoses in that region. Since the cause of atopic dermatitis is food allergy, the treatment is to discover and eliminate the offending food or foods. The local treatment of the skin lesion is usually the application of saline or weak bicarbonate compresses to the oozing lesions and a 2 per cent tar ointment to the dry lesions.

Perianal dermatitis can be differentiated from the other diaper dermatoses by its clinical characteristics:

1. It involves the perianal area and is due to stool irritation.
2. Usually it is most inflamed about the anus and fades in the periphery.
3. It occurs most commonly in the newborn infant, subsiding spontaneously in two or three weeks.
4. It is observed in children and adults following oral antibiotics and may persist for several weeks or months.
5. It is observed following diarrhea from any cause, subsiding when the diarrhea has stopped.

The treatment and prevention of perianal dermatitis are best accomplished by protecting the skin from the irritating stools. After every stool the buttocks should be flushed with lukewarm

water and gently patted dry with a cotton pledget. The skin may then be exposed to the air or heat lamp for 15-20 minutes and then covered by a protective cream. The ointment used in this series of perianal dermatitis has proven to be an ideal adjuvant to the treatment. It is easy to apply and maintains an excellent coating to the sensitive skin. It has proven to be efficacius in prevention and treatment, even when no other therapeutic measures were employed.

SUMMARY

1. Perianal dermatitis is a common eruption seen in newborn infants from transitional stools and in children with diarrheal stools, whether infectious or following oral antibiotics.

2. Other dermatoses of the diaper area which are confused with perianal dermatitis are described.

3. Local application of an antibacterial water-repellent and cod liver oil cream with a casein competitive substrate (Diaparene® Peri-Anal) is an efficient and safe agent in the prevention and treatment of perianal dermatitis.

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OBITUARY

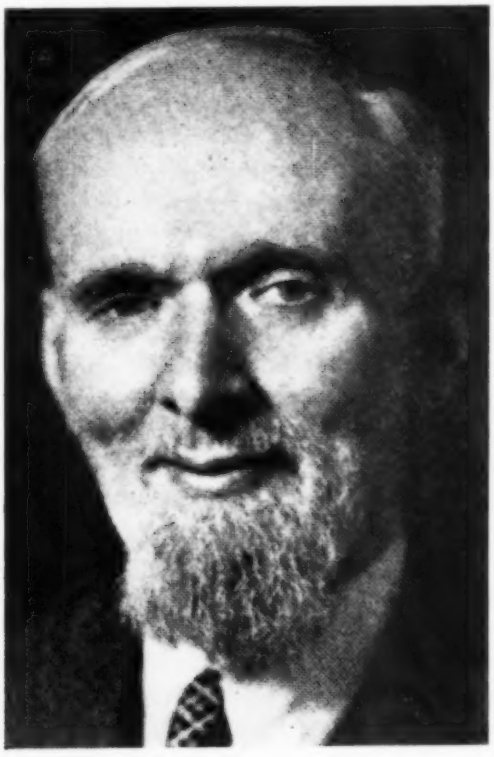
EVERT GORTER
1883-1954

Evert Gorter, professor emeritus of pediatrics in the University of Leyden, an honorary member of the American Pediatric Society, died of neoplastic disease in February 1954. Thus ended the career of a most extraordinary man. His talents as a clinician, a scholar and an organizer brought the pediatric clinic at Leyden to the forefront of European and, indeed, of world pediatrics. His many clinical contributions touched on nearly every phase of pediatrics—from infectious disease to cardiology, kidney disease and nutrition. His written papers are models of clear thinking and simple exposition. He found time to write a textbook and to serve as editor of the Dutch pediatric journal—Maandschrift voor kindergeneeskunde, and of Acta paediatrica. Quite apart from his achievements in pediatrics he was a physicist of note, and over the years made many studies of the behavior of monomolecular films. Most of these studies were undertaken to interpret biological phenomena—the structure of body proteins and lipids. He also devised a practical microanalytical method for blood lipids, based on mononuclear spreading.

In the early 1940's Dr. Gorter was invited to take over the chair of pediatrics at Ghent, in Belgium, and to reorganize the pediatric clinic there. This he was able to do without curtailing any of his activities in Leyden. For some years he commuted three days a week to Ghent. The clinic there was modernized, clinical investigation was started, as well as a laboratory for studying monomolecular films.

In the late 1930's Gorter fell a victim to chronic arthritis in a severe form, and the last 15 years or more of his life were spent in a wheel chair from which he continued to carry on all his activities without interruption. With the invasion of Holland in World War II, he was removed from his post at the University and sent to a small town in the north of Holland to practice pediatrics, a supposedly innocuous post from the point of view of the invaders. As a cripple, little attention was paid to him by the

authorities, an immunity which enabled him to organize and administer a large part of the underground resistance in his country. Throughout this trying period he continued to practice from his wheel chair and, with the aid of a medical student, and with a kitchen sink as his only laboratory, to continue his studies on monomolecular films. At the close of the war he returned to his post at Leyden, a post which he held until his retirement a year ago. Many Americans will remember his participation in the International Pediatrics Congress in New York in 1947.



Professor Dr. Evert Gorter

Dr. Gorter exercised an important influence on pediatrics on both sides of the Atlantic. He was a quiet, modest man of simple tastes, a man of few words, but the quality of whose intellect was readily appreciated. His life was a triumph of mind over matter,

and no small measure of the achievements of his later years is due to his devoted wife and secretary, Mrs. Elizabeth Gorter-Muller, who survives him. He had many friends among the American pediatricians and will be greatly missed.

L. EMMET HOLT, JR., M.D.

MIGRAINE IN CHILDREN. (Maandschrift voor Kindergeneeskunde, Leyden, 21:259, Aug. 1953). In the first part of this report de Wit is concerned with 74 children who had periodic attacks of headache and a history of one or several of the following factors: concussion of the brain, nasal adenoids, disorders of the nasal sinuses, visual defects, and allergic manifestations. Fifteen of these 74 children had family histories of migraine, and 6 of these 15 had allergic disorders. This incidence of allergy is not much higher than what can be expected in the absence of a family history of migraine. Gastrointestinal symptoms, with or without headaches, were observed in 4 of the 15 children with a family history of migraine, and all 4 belonged to the allergic group. In the second part of this paper the author reports observations on 60 children belonging to 26 families with a history of migraine. Fifteen of these children had attacks of migraine before the age of puberty, but only 5 of the 15 retained their migraine attacks during puberty. Eight of the 45 children, who had been free from migraine during childhood, had their first attacks of migraine during puberty. Generally the attacks of migraine were the severer and more frequent, if the onset had been early. Prepuberal migraine occurred with the same frequency in both sexes, whereas more girls than boys had attacks of migraine during and following puberty. All children who had migraine attacks before puberty had a family history of migraine. Allergy does not play an important part in prepuberal migraine; the attacks differ from those in adults only by their greater frequency. Treatment with antihistamines proved most effective in children with migraine, and it appeared that the sedative effect of some of these drugs was not the decisive therapeutic factor. Early treatment is important.—*J.A.M.A.*

PEDIATRICS AT THE TURN OF THE CENTURY

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

HERPANGINA

JOHN ZAHORSKY

In the medical literature of 1915 a reference to an herpetic disease of the throat attracted my attention, since I had seen a few cases of herpetic sore throat that summer. From this experience a clear picture of the eruption on the pharynx remained in my mind. The next summer I saw several more cases and these were reported. (Herpetic Sore Throat, South. M. J., 1920). The following summer an epidemic of this affection, with its uniform symptomatology, tempted me to name the unrecognized disease to which I gave the name of herpangina (a specific infectious disease). (ARCH. PEDIAT., 41: 181, 1924).

The clinical description was incorporated in my book, "Synopsis of Pediatrics," from which this transcription is taken:

"The name is restricted to a specific disease, the lesions of which appear chiefly in the posterior part of the mouth and throat. The disease usually arises in the summer months in an epidemic form: young children are most frequently affected. A strain of the Coxsackie virus has recently been etiologically implicated.

"The disease begins suddenly as an acute febrile movement. The temperature often rises to 104° F. and a convulsion may occur; vomiting may be severe. Anorexia and prostration are sometimes marked. The throat and posterior part of the mouth show minute vesicles surrounded by an inflammatory areola, or, if these have ruptured, small punched-out ulcers. They occur on the anterior pillars of the fauces, the tonsils, the pharynx and the edge of the soft palate. The number of lesions varies from two to twenty. Dysphagia is sometimes marked (in older childern). The general and local symptoms disappear in a few days. The disease may easily be confused with ulcerative stomatitis, which sometimes

begins in the throat. The prognosis is favorable and the treatment is symptomatic."

For thirty years no one seems to have recognized this syndrome, although hundreds of such cases must have been seen by practitioners. It was only a "sore throat," and easily cured by symptomatic remedies. It seems that diseases which run a favorable course are not reported. What about our out-door clinics? But I must not be sarcastic for Levine et al. did publish a report of an epidemic: "An Unusual Epidemic of Possible Herpetic Origin." Also Breese reported his observations on aphthous pharyngitis.

Not until the thorough study of this throat disease by R. H. Huebner et al. (J. A. M. A., March 3, 1951) was any progress recorded. Their studies were made at the Microbiological Division of the National Institutes of Public Health, Bethesda, Maryland, and they conclusively proved that a strain of the Coxsackie virus was the causative agent (The Importance of Coxsackie Virus in Human Disease. New England J. Med., Aug. 1952). Now the name herpangina has been generally adopted, and practitioners everywhere became interested in this common throat infection.

PETROLATUM AS A MEDICAMENT

JOHN ZAHORSKY

Occasionally the practitioner has to fight against certain erroneous practices which are considered correct. The use of mineral oil steadily increased for many years as an internal and external protecting agent. After World War I camphorated oil was made with mineral oil instead of olive oil and this was given hypodermically in many cases of influenza pneumonia. Hard masses were produced under the skin which, after weeks were not absorbed and caused considerable irritation. Then we plainly learned that mineral oil was a foreign substance. I quote from my article "Petrolatum as a Medicament" (ARCH. PEDIAT., May 1933.):

"Long ago we learned that petrolatum was not absorbed when injected parenterally and it remained in the tissues as a foreign body around which a veiled chronic inflammatory reaction developed after a varying interval. Liquid petrolatum is not saponifiable like the true fats; the human body can do nothing with it

except to encase it in a fibrous coating. True, when the deposit is near the surface, as in the mucous membrane, the leukocytes and giant cells may gradually convey it to the surface. Then we learned that liquid petrolatum when aspirated into the lung produces a singular form of chronic inflammation, now known under the name of liquid pneumonia, the pathology of which is characteristic."

Then I asked and tried to answer: Does the instillation of liquid petrolatum into the nose do good or harm? May not adenoid hypertrophy be increased by this oil? I think it may.

And the absorption of vitamins is diminished by swallowing daily this oil!

My own observation indicated that mineral oil will leave patches of skin disease if it is applied daily on the skin.

The oxide of zinc ointment years ago was made with lard. Lately petrolatum has been substituted and thereby this old remedy for eczema has been vitiated. (The XIII edition of the U. S. Pharmacopia does add a little lanoline to the salve.)

Then Stryker found that "small amounts of petrolatum are absorbed from the alimentary in human subjects and experimented animals."

So the pediatrician can imagine some deficiency in nutrition sooner or later. The lymph glands become clogged with the oil.

I imagined several hundred crypts of Lieberkuhn occluded with droplets of oil. Then ulcers arise in the colon when this oil is administered for a prolonged period (several cases cited).

Summary: "Petrolatum is a foreign body, to be used as an emollient on a sound skin (for protection) and like all foreign matter should be frequently removed with soap and water. Its internal use on the respiratory tract, or in the alimentary tract, is contraindicated. Its harmful influence is so insidious it is generally overlooked."

What good did the article do? I don't know, but I infer that the use of petrolatum as a medicament has enormously declined. I was only a soldier in the combat.

COMMUNICATION

In the April 1953 issue, the Archives published an article by W. J. McCormick, M.D. "Domestic Water Fluoridation." His thesis has aroused considerable comment. The following communication presents an analysis of his data from a dental viewpoint.—The Editor.

To the Editor:

The dental benefits which accrue from ingested water fluorides at a concentration of approximately 1 ppm F have been well documented.[1-3] This documentation is based on studies from areas where fluorides occur at the optimal concentration naturally and from studies where the fluoride ion concentration has been supplemented to bring it up to desired concentration. These studies indicate that if 1 ppm fluoride ion is present in the domestic water supply and is ingested from early infancy on, the dental caries experience will be about ⅔ less than if the water were fluoride deficient. It has also been established that this protection persists through adult life to at least age 44.[4]

Likewise, the safety of ingesting water fluorides at the recommended dosage has been studied and has been attested to by all of the leading agencies and organizations concerned with the health of the public.[5-11]

In a paper appearing in the ARCHIVES OF PEDIATRICS, April 1953, Dr. W. J. McCormick has chosen to ignore all of this positive information concerning the efficacy and safety of water fluoridation as a public health procedure to control dental caries. Instead, he has presented some material, much of it out of context, that appears to favor his preconceived ideas. As I shall demonstrate below, many of Dr. McCormick's references are incorrectly quoted, and others have no bearing on the question of domestic water fluoridation. The abstracting of his paper in the Journal of the American Medical Association,[12] and in the Journal of School Health,[13] has given his ideas wide circulation, and consequently I feel that an answer pointing out these errors is warranted.

McCormick starts his presentation with the premise that dental caries is an index of nutritional status and then goes on to elaborate on the dangers of trying to control this disease with ingested water fluorides. In his initial premise he ignores all of the work which has been done during the past fifty years and which has led to the overwhelming acceptance of the fact that the relationship of dental caries to nutrition is not one of a nutritional deficiency. Rather it is initiated by the end product of fermentable carbohydrate in the mouth. No evidence has been reported that clearly

shows that deficiencies of vitamin C or members of the B complex lead to increased susceptibility to tooth decay,[14] or that additions of these nutrients to diets would decrease the caries susceptibility. Indeed, some surveys indicate that lowered susceptibility to dental caries occurs in the presence of single or multiple deficiencies of the B complex and vitamins A and C.[15] The validity of the study by Hanke et al,[16] cited by McCormick to show the value of vitamin C, is solely dependent on the ability of the examiners to evaluate the progress of decay and the current activity of the carious lesion at each examination and for which no criteria exist.

The work of Nizel and Harris[17] on the relative carogenicity of New England and Texas foods fed to hamsters is offered to provide an alternative nutritive explanation to the protective effect of fluoride on caries in Texas. The results of this study were preliminary and inconclusive. However, contrary to McCormick, riboflavin was not effective in preventing caries in this work, because it was the New England diet that had the higher riboflavin content. It might be noted with interest that Dr. Harris recently endorsed fluoridation of water supplies as the most effective way to reduce dental decay on a large scale.[18]

With regard to the potential toxic effects of ingested fluorides, the paper cites the deleterious effect on cattle grazing in the vicinity of aluminum plants and on humans who work in cryolite mines. It does not indicate the high concentration of fluorides ingested in these cases and leads the unsuspecting to believe these results will occur from drinking water at 1 ppm F. According to Roholm,[19] whose monograph on fluorine intoxication is a classic, the threshold dose of fluorine for cattle is 2-3 mg per kg of body weight. Therefore, a 1,000-pound cow could safely ingest 910-1,365 mgs F daily.

In the cryolite mines Roholm indicated that the miners evidencing spontaneous osteosclerosis caused by ingestion of cryolite dust were getting 0.2-1.0 mg F./kg body weight daily. Therefore a man weighing approximately 75 kg (165 lbs.) ingested from 15-75 mgs F. daily. In water fluoridation we are talking about a child ingesting approximately 1 mg daily. A child weighing 15 kg (32 lbs.) drinking a quart of water with 1 ppm F. would therefore be getting about 0.07 mg/kg of body weight daily.

The "very small amounts" of sodium fluoride that Brandl and Tappenier[20] fed to dogs for 21 months amounted to 402.9 grams or 230 grams in 12 months. This is equivalent to the amount of fluoride that would be ingested by a person drinking 600 quarts of water containing 1 part per million fluoride daily for one year.

Dr. McCormick cites the work of Loevenhart and Pierce[21] (without giving the reference) to show the effect of fluoride on body enzymes. This work, published in 1906, indicated that sodium fluoride as 1 part in 5 million (not in 15 million as stated

by McCormick) would inhibit liver lipase activity. However, pancreatic lipase activity was actually accelerated by the same concentration of fluoride. These erratic results, coupled with the fact that these results have not been verified in all these years, would therefore appear to be without meaning.

The theory of Stuber and Lang[22] that there is a direct correlation between blood fluoride level and prolongation of blood coagulation time was discarded many years ago,[23] particularly since their overall theory of blood coagulation denied the role of thrombin in the process, an untenable position today.

McCormick in his paper states that Dr. H. K. Box, a periodontist of international repute of Toronto, informed him that he had checked the dental status of a number of subjects who had been using naturally fluoridated water for several decades and found "appalling periodontal disease, with marked morphological changes, such as enlargement of the roots and narrowing or closure of root canals and pulp chambers, indicating premature aging of tooth structure, making extraction very difficult and prone to root fracture. Many of these cases had severe gingivitis and pyorrhea and premature need for dentures."

I wrote to Dr. Box to confirm this statement and on April 8, 1954, Dr. Box[24] advised me that McCormick must have misunderstood him because he had never made a survey of periodontal or pulp conditions in any area of naturally fluoridated water. He had discussed with McCormick the work of Roholm who had reported on cryolite intoxication.

I checked further into this question of periodontal disease and morphological changes in teeth resulting from ingested water fluorides with Dr. F. S. McKay in Colorado Springs (2.6 ppm F. in domestic water for 75 years). Dr. McKay is a periodontist of note and a pioneer in fluoride investigations. His reply dated February 7, 1954,[25] states in part: "I cannot characterize his (McCormick's) assertions as to conditions he ascribes to the use of fluorized water in any way other than as utterly and completely unsupported in my own experience and observation. No such relation between the use of our domestic water (2.6-2.8 pp. fluoride) exists in the tissues as set forth in his article among the inhabitants of this city. . . . But not contented with my own observations I have delayed my reply to you until I could submit McCormick's statements to Dr. Wilton W. Cogswell, Sr., of this city. He is a practicing exodontist and oral surgeon here for the past 34 years, an excellent pathologist and author of a textbook on Exodontia. He dismisses McCormick's statements as entirely unfounded and that in his own library of 35,000 cases of dental radiographs he has found nothing resembling what McCormick describes. His opinion is even more emphatic than I have indicated. I know of no one here more capable of passing judgment

in this matter than Cogswell. He states further that the morphology of tooth roots is in no wise different than that among nonfluoride individuals contrary to McCormick's statement."

In a study of gingivitis among school-age children of three communities in Canada recently published,[26] it was concluded that there was no significant relationship between the presence of fluoride in the water supply and the occurrence of gingivitis.

The work of the Smiths[27] is cited to show that the protection against dental caries provided by water fluoridation is largely lost after 21 years of age. Here again, both McCormick and Smith neglect to consider the quantitative aspects of water fluoridation. The data here refer to a small community in Arizona with artesian well supplies providing water with high fluoride concentrations. In the paper cited the concentrations are given as 1.6 to 4.0 parts per million but in a later one by the same authors[28] the values are 3.8 to 7.1, a disturbing inconsistency. All residents of this community had severely mottled teeth. The problems associated with the fragility, esthetics and restoration of such teeth are well known, but are completely irrevelant to the consideration that water containing one part per million fluoride provides caries protection without any disfiguring of teeth.

The study by Russell and Elvove of the prophylactic effect of ingested water fluorides on adults was cited earlier.[4] This controlled study shows conclusively that the dental benefits last at least through age 44. The DMF rates were 60% lower in Colorado Springs compared with Boulder City (fluoride free). The Boulder City adult residents lost three times as many teeth as did those in Colorado Springs.

The latest report on the safety of water fluoridation was made by the Commission on Chronic Illness at its meeting in Chicago March 18, 1954.[29] The report was based on a study by a special committee consisting of Dr. Kenneth F. Maxcy, Professor of Epidemiology, Johns Hopkins University School of Hygiene and Public Health, Baltimore, Dr. Edward J. Stieglitz, outstanding geriatrician of Washington, D. C., and Dr. Nathan Shock, Chief of the Section on Gerontology, National Institutes of Health, Public Health Service. The Commission reported that there was no evidence that the continued consumption of drinking water containing approximately 1 ppm F. is in any way harmful to the health of adults or those suffering from chronic illness of any kind. The report concludes: "The Commission, therefore, urges American communities to adopt this public health measure as a positive step in the prevention of the chronic disease, dental caries."

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INSIDIOUS METATARSAL FRACTURES IN CHILDREN. (Zeitschrift für Orthopädie, Stuttgart, 84:55, 1953). Whereas insidiously progressing metatarsal fractures have long been known to occur in adults and are generally recognized, they have rarely been recognized in children. Popp presents the histories of six children, between the ages of 2½ and 14 years, who had such fractures. The clinical and roentgenologic aspects resembled those of march fracture in adults. It is assumed that these metatarsal fractures in children are not completely due to mechanical factors, but that predisposition, probably on the basis of rachitic or other metabolic disturbances, plays a part.—*J.A.M.A.*

DEPARTMENT OF ABSTRACTS

HARRIS, R. C. AND CAFFEY, J.: CHOLECYSTOGRAPHY IN INFANTS. (Journal American Medical Association, 153:13333, Dec. 12, 1953).

Twenty-seven cholecystographic examinations of 25 patients under 3 years of age were performed with 15 successful visualizations of the gallbladder. Among 12 infants under 6 months of age given priodax or telepaque, visualization was successful in 6. These were given orally in dosage of 0.15 gm. per Kg. of body weight. In 4 patients, double and triple doses were given without toxic effects. Sodium bicarbonate given with priodax did not clearly aid in absorption of the contrast material. Urokon, 70 per cent, given intravenously in dosage of 0.7 gm. per Kg., did not make the gallbladders of 3 infants opaque. The gallbladder shadow was most distinct in the majority between 4 and 9 hours after administration of the contrast material. In 3 patients the gallbladder shadow was seen in only one film out of several. This demonstrates the necessity for taking serial films, in general, earlier in young children than in adults. MICHAEL A. BRESCIA, M.D.

WILLIAMS, H. O'REILLY AND WILLIAMS, A.: FOURTEEN CASES OF IDIOPATHIC MYOCARDITIS IN INFANTS AND CHILDREN. (Archives of Disease in Childhood, 28:271, Aug. 1953).

Idiopathic myocarditis is a serious, frequently fatal, malady occurring usually in the first two years of life. The pathological lesion consists of widespread but usually patchy muscle damage with an associated inflammatory cell reaction. The common clinical illness is a rapidly developing, usually fatal cardiac failure which is manifested by severe respiratory embarrassment, cyanosis, tachycardia with low or normal temperature, an enlarged heart and liver, and electrocardiographic changes of myocardial damage. The illness in some patients may be more insidious in onset and the heart failure not as severe. Recovery frequently occurs after a long illness but there is permanent myocardial damage. The acute form of the disorder is commonly mistaken for pneumonia, as respiratory embarrassment and cyanosis are the outstanding

clinical features, and cardiac enlargement is difficult and frequently impossible to detect clinically. Treatment with digitalis and oxygen has been of very definite value in some patients. The clinical and pathological evidence gives no clue as to the etiology.

AUTHORS' SUMMARY.

JAEGER, R.: CONGENITAL SPINAL MENINGOCELE. (Journal American Medical Association, 153: 792, Oct. 31, 1953).

A study of 68 case histories of patients with all types of spinal meningoceles shows that by far the greatest number of lesions occurred at the lumbar level (75 per cent). Involvement of the cord was uncommon in the upper dorsal and cervical regions. Hydrocephalus was a complication in 40 per cent of the cases. There was bladder, rectal and leg paralysis in 60 per cent of the entire group and in 75 per cent of those with cauda equina lesions. Death occurred before 6 months in 35 per cent, and no meningocele patient with marked bladder dysfunction has reached adulthood. Congenital spinal meningocele with bladder paralysis is a serious disorder. Only a few persons with this disorder can ultimately survive its complications. A conservative attitude is desirable in the treatment of the lesion, since surgical reparative procedures seldom alter the prognosis. Careful psychological preparation of the parents, good general medical management, and long-range, well-planned nursing care are essential to the patient's comfort and health. AUTHOR'S SUMMARY.

MCCORRISTON, L. D.: HYDROCORTISONE (COMPOUND F) ACETATE OINTMENT IN ECZEMA OF INFANTS AND CHILDREN. (Canadian Medical Association Journal, 70:59, Jan. 1954).

One hundred and four infants and children with eczema, with or without a family history of allergy, were treated with 1 and 2½ per cent concentrations or hydrocortisone acetate in various ointment bases. It was found to be 75 to 100 per cent effective in all cases in the proper bases. Improvement occurred within 24 to 48 hours of the beginning of the treatment, and was maintained with therapy and subsequently maintained in most cases for considerable periods of time after cessation of therapy. Any "flare-ups" were much easier to control. In about 60 per cent of the patients there has been no recurrence of the eruption. It was thought to be the

single most effective agent in the treatment of eczema of infants and children. These patients have now been observed over a period of eight months. There are apparently no contraindications to its use. There is probably too little absorption for adverse systemic effects, and no cases of allergic hypersensitivity to date. Its mechanism of action on the skin is unknown. AUTHOR'S SUMMARY.

STEIN, S. C. AND ARONSON, J. D.: THE OCCURRENCE OF PULMONARY LESIONS IN BCG-VACCINATED AND UNVACCINATED PERSONS. (American Review of Tuberculosis, 68:695, Nov. 1953).

The incidence of tuberculous and nontuberculous pulmonary lesions was determined in 1,540 BCG-vaccinated and 1,450 unvaccinated persons. These two groups, negative to tuberculin at the beginning of the study, were comparable in age, sex, economic condition, and in the number of follow-up examinations. The morbidity from tuberculosis was based on serial roentgenograms of the chest taken at approximately annual intervals for a period of 9 to 11 years. Pulmonary lesions presenting the roentgenographic characteristics of primary tuberculosis, minimal, moderately advanced, and far advanced tuberculosis, miliary tuberculosis, and pleural effusion were found in 64 (4.1 per cent) of the BCG-vaccinated persons and in 238 (16.4 per cent) of the control group. Pulmonary lesions, presenting the roentgenographic characteristics of suspected primary, suspected reinfection tuberculosis, nontuberculous lesions, and calcified nodules, were found in 193 (12.5 per cent) of those vaccinated and in 215 (14.8 per cent) of the control group. Primary lesions of tuberculosis were observed most frequently in the first decennium of life, while definite reinfection type tuberculosis and pleural effusions occurred most frequently in the second decennium. Among the control group, the incidence of tuberculous pulmonary lesions was higher in the females than in the males; while, among the vaccinated persons, the incidence of such lesions was the same as, or slightly lower, among the females than among the males. The highest incidence of pulmonary lesions among the BCG-vaccinated persons occurred among those who had received BCG vaccine of low antigenic value. A comparison of the morbidity from pulmonary tuberculosis, as determined roentgenographically in BCG-vaccinated and control persons, gives added evidence of the protective value of BCG vaccine previously

indicated by studies of the rate of mortality from tuberculosis in the same groups. AUTHORS' SUMMARY.

HOLT, J. F.; LATOURETTE, H. B. AND WATSON, E. H.: PHYSIOLOGICAL BOWING OF THE LEGS IN YOUNG CHILDREN. (Journal American Medical Association, 154:390, Jan. 30, 1954).

Exaggerated bowing of the lower extremities occurs with surprising frequency in apparently healthy, well-nourished infants and children. The deformity, usually recognized in the immediate postambulatory period, is generally not due to rickets, osteochondritis, or serious developmental fault. It is thought instead to be the result of delayed resolution of normal, physiological bowing that is present to some extent in all infants. In most instances the prognosis for complete, spontaneous regression of the bowing is excellent, the legs usually having straightened by the age of 4 or 5 years. Roentgenologic examination is imperative to rule out significant metabolic disease, but results of such examination may be misleading. Particular attention is directed to the confusing mechanical structural changes and exaggerated anatomic variants that are the result—not the cause—of the bowing.

AUTHORS' SUMMARY.

HUNTER, O. B.: CORTISONE THERAPY IN RH INCOMPATIBILITIES. (Journal American Medical Association, 154:905, March 13, 1954).

The normal rise of 17-ketosteroid and pregnanediol levels in the urine during pregnancy may be used as a means of following the course of the fetus of the Rh negative mother with a high antibody titer. Administration of cortisone should be started when the normal progression of these hormones falters. Twenty-five mgs. of cortisone every six hours is sufficient at the start. The dosage may be increased if the hormone level does not continue to rise. The newborn child may be expected to lose a small amount of weight. An exchange transfusion is usually necessary to complete the therapy of the newborn infant. This therapy reduced the mortality rate of babies born alive from 10 per cent, with exchange transfusion alone, to 3 per cent. The incidence of stillbirths was reduced from 17 per cent to 5 per cent. AUTHOR'S SUMMARY.

LANMAN, J. T. AND DANCIS, J.: RETROLENTAL FIBROPLASIA AND OXYGEN THERAPY. (Journal American Medical Association, 155:223, May 15, 1954).

In a comparison of liberal and restricted oxygen therapy in premature infants, irreversible, cicatricial retrolental fibroplasia (RLF) appeared in infants in the group with high oxygen but not in the group with low oxygen. Reversible, vascular stage lesions occurred in both groups, but with nine times the frequency in the group with high oxygen concentration. These differences were significant at the 5 per cent and the 0.1 per cent level, respectively. Excluding infants dying of recognized causes with no known relationship to oxygen therapy, the mortality rates in the groups with both high and low oxygen concentrations were the same. We believe that retrolental fibroplasia is directly related to the excessive administration of oxygen and can be controlled by severely limiting oxygen therapy to premature infants. Such restriction does not appear harmful. In the Bellevue Hospital Premature Nursery, oxygen is given only to definitely cyanotic infants, and then at measured concentrations below 40 per cent. Frequent attempts to discontinue therapy are made. Aerosol therapy is given to noncyanotic infants with compressed air.

AUTHORS' SUMMARY.

BRIDGE, R. G. AND FOLEY, F. E.: PLACENTAL TRANSMISSION OF THE LUPUS ERYTHEMATOSUS FACTOR. (American Journal Medical Sciences, 227:1, Jan. 1954).

The lupus erythematosus phenomenon has been demonstrated in the newborn of a mother with disseminated lupus erythematosus. Observations on this situation have not been previously reported. The presence of the lupus erythematosus phenomenon in the baby was considered to be a result of placental transmission of the lupus erythematosus factor. Evidence was equivocal, but suggestive, that the lupus erythematosus phenomenon was still demonstrable in the baby 7 weeks after delivery. It was definitely absent 4 months after delivery. By the methods used there was no evidence of production of antibodies against the lupus erythematosus factor in the baby. The significance of these findings is discussed.

AUTHORS' SUMMARY.

BOOK REVIEWS

THE DYNAMICS OF VIRUS AND RICKETTSIAL INFECTIONS. Edited by Frank W. Hartman, M.D., Frank L. Horsfall, Jr., M.D. and John G. Kidd, M.D. Cloth. Illustrated. Pp. 461. Price $7.50. New York: The Blakiston Company, Inc., 1954.

This book contains the papers and discussions presented at the International Symposium on the Dynamics of Virus and Rickettsial Infections sponsored by the Henry Ford Hospital, Detroit, Michigan, and held at the Hospital, October 21, 22 and 23, 1953. There were some 33 participants from all over the world who presented papers or participated in the discussions. The book is divided in five parts devoted to the following subjects: Mechanisms of Virus and Rickettsial Infections; Ecology and Pathogenesis of Virus and Rickettsial Infections; Mechanisms of Immunity in Virus and Rickettsial Infections; Laboratory Diagnosis of Virus and Rickettsial Infections; and Approaches to Prophylaxis and Therapy of Virus and Rickettsial Infections. Although the papers presented in this volume might be considered ultra-scientific, they nonetheless would be of interest to the discerning clinician in that they relate fundamental knowledge and in turn make clearer some of the processes that occur at the patient level. The papers in Parts three and five were especially fascinating and very instructive. Another paper on the Pathogenic Mechanisms of Virus Diseases was also quite interesting. This book might be considered "heavy" reading for most of us who do not deal with these fundamental problems as a daily regimen. However, a slow perusal of this volume would be rewarding.

MICHAEL A. BRESCIA, M.D.

PEDIATRIC GYNECOLOGY. By Goodrich C. Schauffler, M.D. 3rd Ed., Cloth, Pp. 318. Illustrated. Price $7.50. Chicago: The Year Book Publishers, Inc., 1953.

This book is of great value and contains much information relative to the female genital tract of the infant and child. A specialized book of this nature is an important addition to any physician's library who deals with female infants and children. The text is concise, easily read and the accompanying illustrations in most instances are revealing and informative. The book is highly recommended. MICHAEL A. BRESCIA, M.D.

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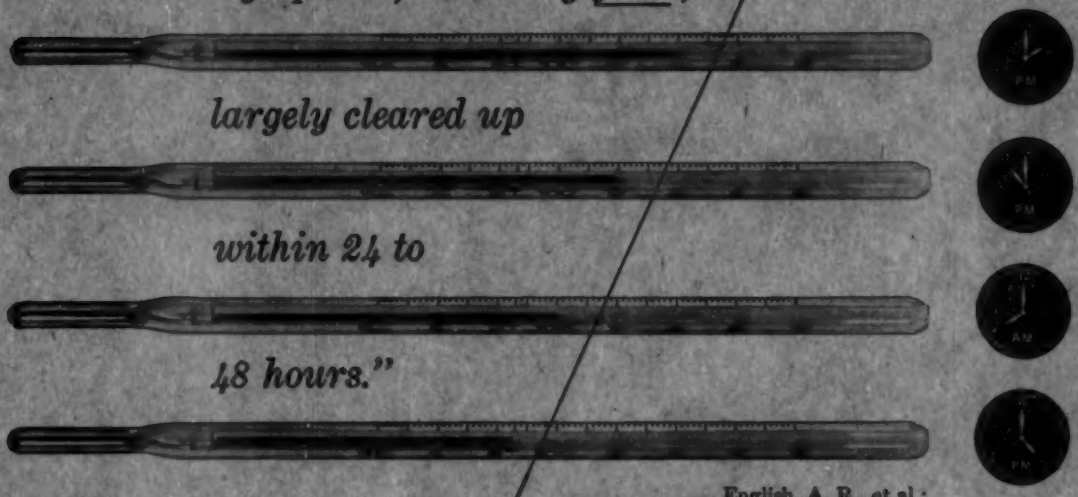

largely cleared up

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English, A. R., et al.:
Antibiotics Annual (1953-1954),
New York, Medical
Encyclopedia, Inc., 1953, p. 70.

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